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Identification of bovine ephemeral fever (BEF) outbreak in a large dairy farm in Varamin, Iran

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Abstract

Bovine Ephemeral Fever (BEF) flared up in a dairy farm with 2097 animals. The disease started in September, 2006, with daily means of environmental temperature (ET) and relative humidity (RH) of 23.8 ºC and 37%, respectively, and ended after 48 days with ET and RH of 16.2 ºC and 68%, respectively. In this outbreak, the age of affected animals was above 10 months and the morbidity rate was 13.07%. Clinical signs included fever, hyperpnoea, mouth breathing, subcutaneous emphysema and death. Histologically, there were vasculitis, hyperemia; hemorrhage and edema in soft tissues and rupture of alveolar walls. Both Culex and Colicoides spp. were captured as vectors. Bovine Ephemeral Fever virus genome was detected in blood samples by RT-PCR and the CPE was shown by blood sample culture.

Keywords: Bovine ephemeral fever, vasculitis, subcutaneous emphysema, pneumoperitoneum

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Introduction

Bovine Ephemeral Fever (BEF) virus belongs to Rhabdoviridae family and Ephemerovirus genus. From time to time even in a single country the viral antigenicity and virulence may change. BEFV has some common antigens with Akabane and Aino viruses (Tzipori et al. 1975; Wang et al. 2001; Andrews et al. 2004; Walker 2005). The first epizootic outbreak of BEF was reported in central Africa in 1867 and nowadays it is a well-known disease in Africa, Australia, Asia and the Middle East. In the Middle East, the disease outbreak has a history from 1931 and in Iran it was first documented in 1974. BEF has become enzootic with epizootic flare-ups in different parts of the world (Shirakawa et al, 1994; Abu Alzein et al, 1999; Nandi and Negi 1999; Walker, 2005; Yeruham et al. 2005; Radostits et al., 2007; Farag, 1998; Taghipour Bazargani, unpublished data).

It is a general belief that insect vectors not only transmit the virus but can maintain it and transmit the BEFV through ovary (Shirakawa et al., 1994; Yeruham et al. 2002; Yeruham et al. 2005). Because of the difficulty of combating with the vectors and shortening the intervals between the outbreaks, different types of vaccines with different efficacy and immunogenic durability have been tested. By now, preparing of G protein vaccine as a pure subunit or recombinant viral vectors is under investigation (Tzipori et al 1975; St George, 1985; Anderson and Mckay,1993; Vanselow et al. 1995; Nandi and Negi, 1999; Wang et al. 2001; Hsieh et al, 2006; Radostits et al, 2007).

Materials and methods

During the BEF outbreak, different factors including weather, age, sex, productive and reproductive status, and treatment outcome of each BEF affected animal were ascertained. In addition, in some dead or emergency slaughtered animals macroscopic lesions were recorded and then, tissue specimens of lung, heart, liver, lymph nodes and skeletal muscle were fixed in 10% buffered neutral formalin. After routine tissue processing, 5-6 µm tissue sections were stained with Hematoxylin and eosin and evaluated histologically. BEFV was detected in blood samples of some pyretic animals by cell culture and Polymerase chain reaction (PCR) techniques. Moreover vector insect was captured and specified. From epidemiological point of view, stables' characteristics, locations and designs including inside and outside lightening and animal distribution in stables were evaluated by a civil engineer. Percentage of BEF affliction in sections of each stable was calculated. Data were analyzed by SPSS (Ver. 11) using Chi-square and Fischer's exact tests.

Results

Epidemiology

BEF flared up in a commercial dairy herd containing 2097 animals. The disease appeared in September 2006. The average of environmental temperature (ET) and relative humidity (RH) were 23.8 ºC and 37% at the occurrence time of the disease and 16.2 ºC and 68%, respectively when the disease disappeared. During this period, 274 animals were affected and the lowest age of affected animals was 10 months old. The morbidity rate of the total population and at risk animals were 13.37% and 20.68% respectively. Frequencies of affection during each of the three consecutive 16 days of the disease were 32.8%, 43.6% and 23.36%. The difference in percentage of afflictions between non-productive cows and bulls on the one hand and pregnant heifers and productive cows on the other hand as well as in between productive animals were significant (p=0001), but without any steady trend (Tables 1 and 2). As it is shown in figure 1, the percentage of affected cows in the first and second parities had significant (p=0.005) increment trend, but there was no steady trend in the remainder of parities. The figure 1 also shows that the percentage of BEF occurrence in newly calved

cows was significantly (*p*=0.0001) higher than that of high producing cows. Low milk producing cows (LMP<20L) had significantly (*P*=0.001) more incidence than high milk producing cattle (HMP> 40L).

Vectors

Most of captured mosquitoes were Culex (70%) and Culicoides (20%) and the remaining were plant insects. Culex mosquitoes were belonged to two species, C. theilery and C. pipiens.

Clinical Symptoms

Pyrexia was the first clinical sign but 24.54% of all sick animals did not show abnormal body temperature. In pyretic animals, body temperature ranged from 39.5°C to 42°C. The second clinical symptom was hyperpnoea and pulmonary emphysema developing along with the disease progression. Pulmonary emphysema was the most important cause of the dyspnea and even death. Pulmonary emphysema caused non-evenly distributed subcutaneous emphysema in the back, scapular region, neck and upper trunk either in one side or bilaterally in 2.9% of cases. In some cases, subcutaneous emphysema as well as pneumoperitoneum was seen. In spite of having muscular and articular pain, affected animals did not tend to rest even on a thick bed of straw. These animals showed mouth breathing with a protruded tongue and had abundant foamy discharge in the mouth. Affected animals became anorexic very soon. Sticky nasal discharge, hypersalivation with foamy and sticky saliva and depression were signs that animal keepers detected the BEF affected animals immediately.

During the outbreak, abortion and stillbirth rates increased at the rate of 48% and 1.14%, respectively.

Necropsy findings

Pulmonary emphysema with different severity was present in nearly all died or slaughtered animals. In a case with subcutaneous emphysema in the neck and back areas almost all parts of the lung were affected. In this case, the right lung was 62 cm and its diaphragmatic lobe was 30 cm while the above-mentioned diameters for left lung were 62.22 and 34 cm, respectively. Pericardium and connective tissues around the spinal cord from thorax to the lumbar region of the animal were emphysematous too. There was also severe epicardial hemorrhage.

Microscopic findings

Histopathologic examination revealed pulmonary hyperemia, vasculitis (Fig. 1), emphysema and atelectasis (Fig. 2). In the liver, there was moderate hyperemia with neutrophilic infiltration in sinusoids. There was also acute lymphadenitis. Necrosis of rhabdomyocytes with hyperemia and hemorrhage was notable (Fig. 3). Cardiac muscles showed hyperemia and extensive sub-endocardial hemorrhage (Fig. 4).

Virus culture and Polymerase Chain Reaction test

Blood samples of clinically BEF affected animals were immediately referred to the virology laboratory, Razi institute for vaccine and serum development, Karaj, Iran. Buffy coats were injected intracerebrally into the brain of 2-day-old mice. After 5 days, signs of posterior paralysis were seen in mice and then the virus was passaged for the second time. Then, brain infusions were inoculated to Vero and BK.RAZI cell cultures. Following the appearance of minor CPE, the cultures were frozen, thawed and passaged for 5-6 times until definitive CPE was detectable (Fig. 5).

After isolation of viral RNA from buffy coats of whole blood and preparation of cDNA, polymerase chain reaction test was carried out on the samples and the presence of the virus was approved by specific amplification of 840bp fragment of BEFV genome (Fig .6). The following primers were used: Forward (12):

5'-CCTAATAATTACCTTATTAGTCAATG-3'

Reverse (1614):

5'-CCCTAATTCCAAATCTTGATAATTCT-3'

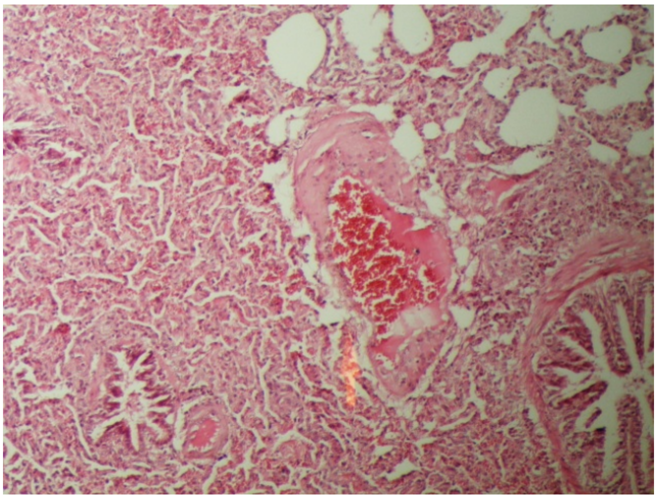


Figure 1. Vasculitis in the lung. Hematoxylin and eosin,×100

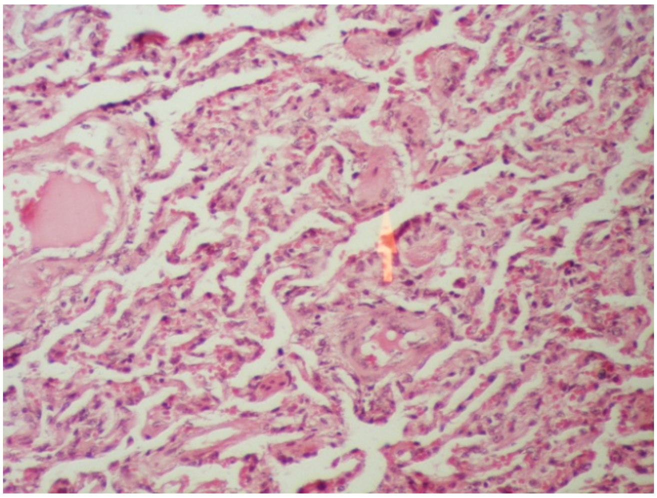


Figure 2. Acute interstitial pneumonia with smooth muscle hyperplasia in lung. Hematoxylin and eosin,×200

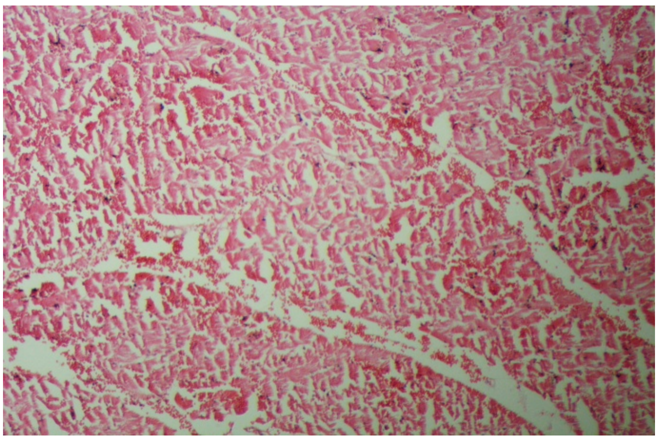


Figure 3. Extensive skeletal muscle necrosis and hemorrhage. Hematoxylin and eosin,×200

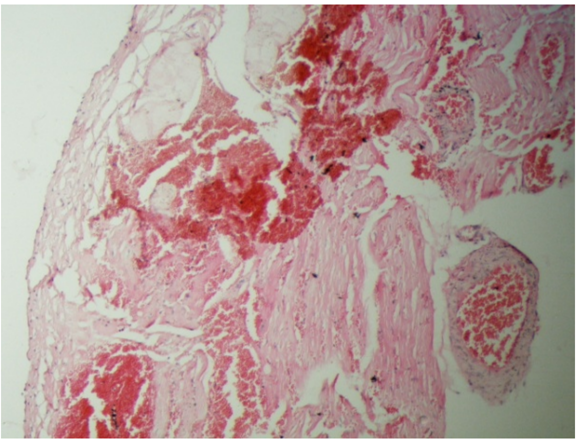


Figure 4. Extensive subendocardial hemorrhage. Hematoxylin and eosin,×100

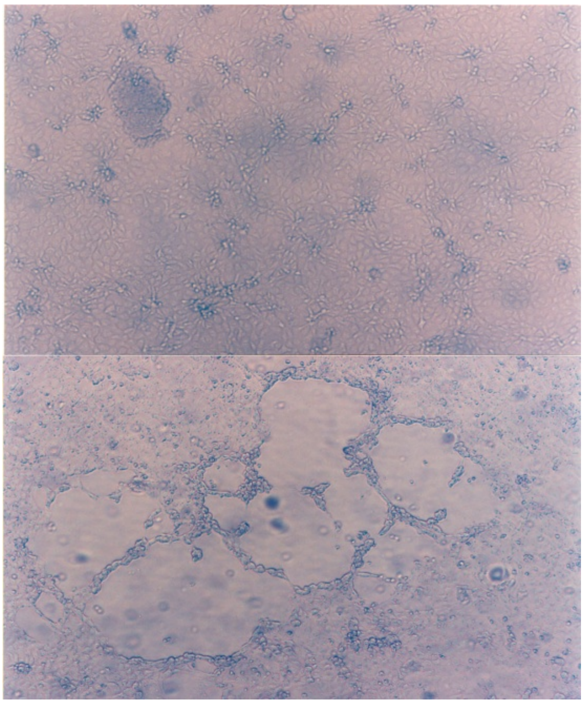


Figure 5. CPE of the BEFV in cell culture (down) compared to the normal cell culture (up).

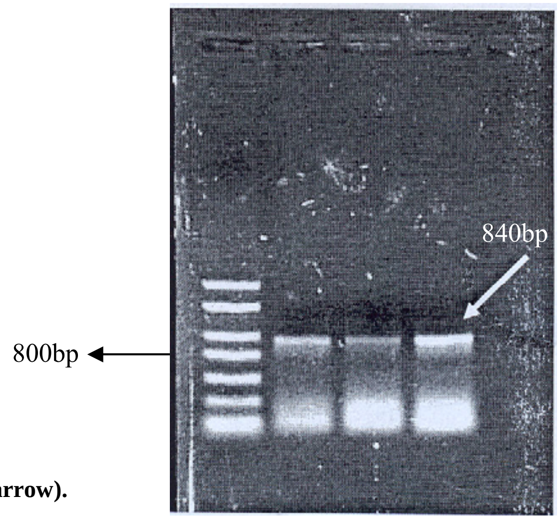


Figure 6. PCR amplification product.
The 840bp specific fragment of BEFV genome (arrow).

Treatment

All the affected animals were showing pyrexia received flunexin meglumine (2ml/45Kg/daily, IV) and ceftiofur (1ml/Kg/daily,SC). Vitamin B-complex (3.5mg/50Kg/daily, IM) and vitamin E-Selenium (5-10ml/head, SC) were injected to 79.5% and 17.15 % of BEF affected animals respectively. Thick straw beds were prepared for downer animals. In addition, triplenamine hydrochloride (0.5mg/Kg/D, IV) was injected downer animals as a central nervous system stimulator. Fluid therapy was considered in order to supply energy, water and electrolytes for anorexic animals In cases with anaphylactic shock, adrenalin (0.02 ml/Kg, IM) and/or dexamethasone (0.1mg/Kg, IV) were used. To reduce the volume and distribution of subcutaneous emphysema, skin incisions were made on the back of affected animals.

In spite of all these treatments, 18.64% of the affected animals died (9.83%), slaughtered (23.5%) and sold alive (66.67%). Culling percentage in each of 3 consecutive 16 days periods were 35.29%, 37.26% and 27.45%, respectively. Culling rate of the affected cows and other at risk animals were 18.25% and 0.36% respectively. The difference between these two classes of culling was statistically significant (p=0.0005). Vaccination of at-risk animals started from July 2007.

Discussion

The first outbreak of BEF in Varamin district was recorded in 1981 (Bazargani TT, unpublished data). Since then BEF has been occurred either in mild to severe enzootic forms or in epizootic form flaring up not only in this district but also in some other areas nearby or even quite far from Varamin. The two latest BEF outbreaks in Varamin occurred in 2002 and 2006. This is one of the characteristics of the BEFV that tends to become endemic in an area (Yeruham *et al*., 2002).

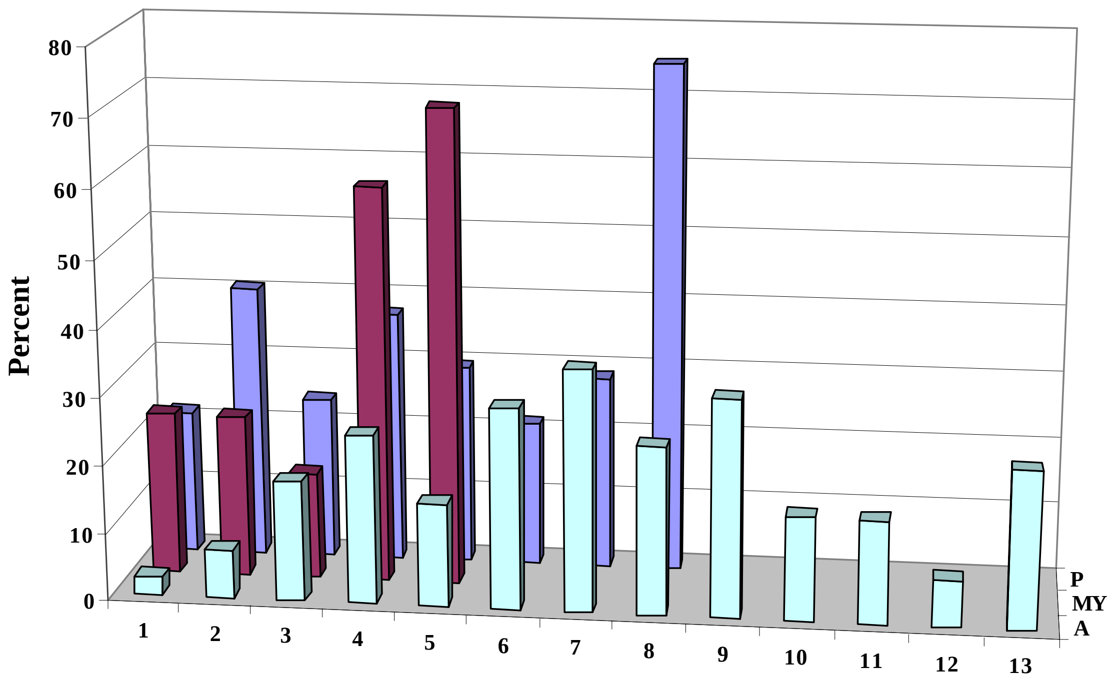
Culex and Cullicoid insects were caught in the last outbreak at this dairy farm. It seemed that the external light projectors significantly increased the rate of affection. Culex mosquitoes are night haematophagus and night light stimulates their activity (Soulsby, 1982), it was concluded that Culex mosquitoes were the most important vectors in this dairy farm. Control of the vectors by using of insecticides appeared nearly impracticable and the net economic cost caused by the BEF outbreak in Varamin district was very threatening for dairy industry.

Based on the results of this study, it seems that animals younger than 10 months old were not clinically affected by BEF. This is contrary to the fact that even newborn non-immune calves can experimentally develop the disease (Tzipori, 1975). Meanwhile a study showed that immature calves produced serum neutralization antibodies 1.9 times lower than mature cows (Hsieh *et al.*, 2005). It can be said that in the presence of well located external light projector, desirable environment for vector egg deposition and hatching condition, height of animals, and stressful conditions might have been responsible for the outbreaks of the disease in this farm. The epidemiology of this BEF outbreak (Table 3) was very similar to the other countries in the Middle East. Moreover vector population and their seasonal activity had the same pattern in this region (Yeruham *et al*., 2002; Abu elzein *et al*., 2002).

It was claimed that in BEF affected animals, development of emphysema is a reflection of selenium deficiency (Radostits *et al*., 2007). In the present study, even calves aged over three days were received supplements including vitamin E and Selenium. In order to reduce the rate of placental retention during the dry period, cows were administered vitamin E-Selenium solution. Therefore, the subcutaneous emphysema which were seen in two affected dry cows could not be due to selenium deficiency.

Because fever in BEF shows polycyclic shifting patterns in 24 hours (Grant Maxie, 2007), 24.54% of non-pyretic BEF afflicted animals might have been monitored in one of those shifting periods.

It has been reported that in BEF affected herd the incidence in bulls were higher than cows (Yeruham *et al*., 2003). In the present report, none of beef cows equal or older than 14 months as well as bulls showed any signs of the disease. This obvious contradictory finding clearly indicates that the rate of affection in BEF is not just dependent on the sex, age and weight of at risk animals.



	1	2	3	4	5	6	7	8	9	10	11	12	13
Age (m)	6 12	13 19	20 26	27 32	33 39	40 46	47 53	54 60	61 67	68 74	75 81	82 88	89 95
Milk yield	Low	Medium	High	High	N.C								
Parity	1st	2nd	3rd	4th	5th	6th	7th	8th					

Diagram 1. BEF affection rate in different age, milk production and parity groups.
P=parity, MY=Milk yield, A=age

Table 1. BEF incidence rate in non-productive female and male animals and in bulls.

age	gender						
	female			male			
	total	%affected	%total affection	total	affected	%total affection	%to total affected
0-3M	128	0	0	117	0	0	0
3-4M	48	0	0	48	0	0	0
5-7M	48	0	0	48	0	0	0
8-11M	68	2	2.8	60	0	0	1.56
12-14M	55	4	7.27	110	5	4.54	7.8
>14M	-	-	-	40	0	0	0

Table 2. BEF incidence rate in heifers and in productive cows

parity	Total No	Affected No	% affection	% to total affceted
Heifer NP	123	13	10.53	4.74
Heifer P	150	32	21.33	13.41
1rst Pa	329	70	21.27	25.54
2nd Pa	195	79	40.5	28.83
3rd Pa	134	32	23.88	13.41
4th Pa	59	22	33.2	8.2
5th Pa	27	8	29.62	2.91
6th Pa	14	3	21.42	1.09
7th Pa	7	2	28.5	0.72
=/> 8th Pa	4	3	75	1.09

Table 3. Varamin district weather during 2002-2006

Year	2002			2003			2004			2005			2006		
Mo.	Sept	Oct	Nov	Sep	Oct	Nov	Sep	Oct	Nov	Sep	Oct	Nov	Sep	Oct	Nov
Temp.	27.9	23.7	14.4	20.9	21.4	14.3	26.6	21.9	15.	27.3	23	12.6	26.4	22	14.8
RH%	33.0	31.5	49.0	31.5	33.0	49.5	40.5	37.5	52.5	38.5	41.5	58.0	35.0	45.0	55.0
Wind Direction	S W	N W	N W	N W	N W	N W	S E	N W	W S	N W	N W	N W			

References

Abu Elzein EM, Gameel AA, al-Afaleq AI, al-Gundi O, al-Bashier AM, Zeedan A, al-Mageed HA, Abu Khadra H.(1999) , Observations on the recent epizootic of bovine ephemeral fever in Saudi Arabia. *Revue scientifique et technique* **18**, 672-80.

Bai WB, Jiang CL, Davis SS. (1991) Preliminary observations on the epidemiology of bovine ephemeral fever in China. *Tropical Animal Health and Production* **23**, 22-26.

Bazarghani TT, Charkhkar S, Doroudi J and Bani Hassan E. (2006) A review on Pest des Petits Ruminants (PPR) with special reference to PPr in Iran, *Journal* of *Veterinary Medicine* B **53**, 17-18.

Burgess GW, Spradbrow PB. (1977) Studies on the pathogenesis of bovine ephemeral fever. *Australian Veterinary Journal* **53**, 363-8.

Chang C. J., Shih W.L., Yu F.L., Liao M.H., Liu H.J. (2004) Apoptosis induced by bovine ephemeral fever virus, *Journal of Virological Methods* **122**, 165-170.

Elamin MA, Spradbrow PB. (1978) Isolation and cultivation of bovine ephemeral fever virus in chickens and chicken embryos. *Journal of Hygiene (Lond)* **81**,1-7.

Farag MA,al-Sukayran A, Mazloum KS, al-Bukomy AM. (1998) Epizootics of bovine ephemeral fever on dairy farms in Saudi Arabia, *Revue scientifique et technique* **17**,713-22.

George TD, Standfast HA, Christie DG, Knott SG, Morgan IR. (1977) The epizootiology of bovine ephemeral fever in Australia and Papua-New Guinea, *Australian Veterinary Journal* **53**, 17-28.

Hall WT,Daddow KN, Dimmock CK, George TD, Standfast HA. (1975) The infection of merino sheep with bovine ephemeral fever virus. *Australian Veterinary Journal* **51**, 344-346.

Hazrati A., Hessami Ghajar M., Roustai M.H. and Dayhim F. (1974) Bovine ephemeral fever. Proceedings of the 11th Regional Seminar of the National Veterinary Organization, Shiraz-Iran, pp.327-351.

Hertig C, Pye AD, Hyatt AD, Davis SS, McWilliam SM, Heine HG, Walker PJ, Boyle DB. (1996) Vaccinia virus-expressed bovine ephemeral fever virus G but not G(NS) glycoprotein induces neutralizing antibodies and protects against experimental infection. *Journal of General Virology* **77** (Pt 4), 631-640

Hsieh YC, Chen SH, Chou CC, Ting LJ, Itakura

C, Wang FI. (2005) Bovine ephemeral fever in Taiwan (2001-2002). *Journal of Veterinary Medical Science* **67**,411-416.

Hsieh YC, Chen SH, Huang,J-Y, LeeY-F, Tsai K-Y, Liu, H-J. (2005) Virus identification and serum antibody monitoring of bovine ephemeral fever in Tainan county during 2002-2004. *Taiwan Veterinary Journal* **31**, 208-216.

Hsieh YC, Wang SY, Lee YF, Chen SH, Mak PO, Chu CY. (2006) DNA sequence analysis of glycoprotein G gene of bovine ephemeral fever virus and development of a double oil emulsion vaccine against bovine ephemeral fever. *Journal of Veterinary Medical Science* **68**, 543-548.

Kirkland PD. (2002) Akabane and bovine ephemeral fever virus infections. *Veterinary* Clinics of *North America*: *Food Animal Practice* **18**,501-514.

Grant Maxie M. JUbb (2007) Kennedy and Palmer's pathology of Domestic Animals. Vol.3, p83. SAUNDERS (Elsevier)

Nandi S, Negi BS. (1999) Bovine ephemeral fever: a review. *Comparative immunology, microbiology and infectious diseases* **22**, 81-91.

Odiawo GO. (1989) The relationship between selenium deficiency and the development of pulmonary and subcutaneous emphysema in bovine ephemeral fever virus-infected cattle. *Onderstepoort Journal* of *Veterinary Research* **56**,123-125.

Shirakawa H, Ishibashi K, Ogawa T. A. (1994) comparison of the epidemiology of bovine ephemeral fever in South Korea and south-western Japan. *Australian Veterinary Journal* **71**, 50-52.

St George TD. (1985)Studies on the pathogenesis of bovine ephemeral fever in sentinel cattle. I. Virology and serology. *Veterinary Microbiology* **10**, 493-504.

St George TD, Murphy GM, Burren B, Uren MF.(1995) Studies on the pathogenesis of bovine ephemeral fever. IV: A comparison with the inflammatory events in milk fever of cattle, *Veterinary Microbiology* **46**, 131-142.

Tzipori S. (1975) The susceptibility of young and newborn calves to bovine ephemeral fever virus. *Australian Veterinary Journal* **51**, 251-253.

Uren MF. (1989) Bovine ephemeral fever., *Australian Veterinary Journal* **66**(8), 233-236.

Vanselow BA, Walthall JC, Abetz I. (1995) Field trials of ephemeral fever vaccines. *Veterinary Microbiology* **46**, 117-130.

Walker PJ. (2005) Bovine ephemeral fever in Australia and the world. *Current Topics in Microbiology and Immunology*. **292**,57-80.

Wang FI, Hsu AM, Huang KJ. (2001) Bovine ephemeral fever in Taiwan; *Journal* of *Veterinary Diagnostic Investigation* **13**, 462-467.

Tzipori S. (1975) The susceptibility of young and newborn calves too bovine ephemeral fever virus. *Australian Veterinary Journal* **51**, 251-253.

Tzipori TS, Spradbrow PB, Doyle T. (1975) Laboratory and field studies with a bovine ephemeral fever vaccine; *Australian Veterinary Journal* **51**, 244-250.

Uren MF, St George TD, Zakrzewski H. (1989) The effect of anti-inflammatory agents on the clinical expression of bovine ephemeral fever; *Veterinary Microbiology* **9**, 99-111.

Yeruham I, Braverman Y, Yadin H, Van Ham M, Chai D, Tiomkin D, Frank D. (2002) Epidemiological investigations of outbreaks of bovine ephemeral fever in Israel. *Veterinary Record* **151**, 117-121.

Yeruham I, Gur Y, Braverman Y. (2007) Retrospective epidemiological investigation of an outbreak of bovine ephemeral fever in 1991 affecting dairy cattle herds on the Mediterranean coastal plain. *Veterinary Journal* **173**, 192-195.

Yeruham I, Sharir B, Yadin H, Tiomkin D. (2003) Bovine ephemeral fever in beef cattle herds in the Jordan Valley, Israel. *Veterinary Record* **152**, 86.

Young PL, Spradbrow PB. (1980) The role of neutrophils in bovine ephemeral fever virus infection of cattle. Journal of *Infectious Diseases* **142**, 50-55.

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شناسایی وقوع تب کم دوام در یک گله شیری بزرگ در ناحیه ورامین ، ایران

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چکیده

تب کم دوام گاو در یک گله گاو شیری با جمعیت ۲۰۹۷ راس دام بروز کرد. در این گله بیماری با دما و رطوبت نسبی ۲۳/۸ºC و ۳۷٪ شروع و بعد از ۴۸ روز با دما و رطوبت نسبی ۱۶/۲ ºC و ۶۸٪ به پایان رسید. بترتیب ۱۳/۰۷٪، ۲۰/۶٪ کل جمعیت و جمعیت در معرض خطر مبتلا گردیدند. تفاوت درصد ابتلا بین دامهای غیر مولد و مولد و نیز بین گروه های مختلف از دامهای مولد با معنی بود(p<۰/۰۰۰۱).میزان ابتلا رابطه ای با سن، تعداد شکم، و مقدار تولید رابطه ای نشان نداد ولی رابطه آماری محکمی بین وجود پروژکتور _ بعنوان جلب کننده پشه های ناقل – و میزان ابتلا در بخش های مختلف وجود داشت.. در گله مورد مطالعه پشه ها از دسته کولکس و کولیکوئیدس شکار شدند. ژنوم BEF با استفاده از rt-PCT در نمونه خون شناسایی شد و CPE حاصله در کشت نمونه خون مشاهده گردید. تب همراه با افزایش تعداد و عمق تنفس از اولین علامات تب کم دوام بود. با پیشرفت بیماری بعلت آمفیزم ریوی دام خیلی زود شروع به تنفس دهانی می نمود. در همین ارتباط ۲/۹٪ از بیماران آمفیزم زیر جلدی و نیز هوادار شدن صفاق را نشان می دادند و غالباً دام در اثر هیپوکسی تلف می شد. در حالی که هر یک از بیماران مورد نشانه درمانی و درمان پشتیبان قرار می گرفتند ۱۸/۶٪ آنها تلف، کشتار اجباری زنده فروشی شدند. در خلال سه دوره ۱۶ روزه از بیماری نسبت تلف یا ذبح اجتناب ناپذیر از دو راه دیگر بترتیب ۳۵/۲۹، ۳۷/۲۶ و ۲۷/۳۶٪ بود. ۱۸/۲۵٪ از دامهای تلف شده یا حذف اجباری از گاوان بالغ و بقیه از دیگر گروه های بالای ۱۰ ماه بودند. از نظر هیستوپاتولوژیک در غالب موارد پرخونی، خونریزی و ادم بافتهای نرم و نیز پارگی دیواره آلوئولها بعلت واسکولیت مشاهده می شد.

واژگان کلیدی: تب کم دوام، آمفیزم زیر جلدی، هوادار شدن صفاق